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HUMAN RESPONSES TO COLD AFTER REPEATED IMMERSION IN 20°C WATER

by

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The relative importance of core vs. skin temperature reduction as a stimulus for cold acclimation (CA) was assessed by evaluating thermoregulatory responses during cold air exposure (90 min, 5°C; CAE), cold water immersion (60 min, 20°C; CWI), and cold finger immersion (30 min, 4°C; CFI), before and after CA. Subjects acclimated for five weeks by completing daily 1-h water immersions (20°C) while either seated quietly (RG) or performing leg exercise (EG). The exercise intensity for EG was selected to prevent core temperature from falling during each CA session. CFI was completed immediately following CWI (hypothermic condition, HC); and a second trial on a separate day (normothermic condition, NC). Cold-induced vasodilation (CIVD) was not affected by CA, but CIVD was blunted ($P<0.05$) during HC, compared to NC before and after CA. A more pronounced change in NE ($P<0.05$) occurred during CAE following CA, consistent with increased sympathetic activation, which could mediate changes in vasomotor response to cold. The reduction of core temperature during CA sessions appears to be a necessary stimulus for this acclimation effect. This study also suggests that heat debt, not increased heat flux alone, is the stimulus for developing cold acclimation.

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LIST OF ABBREVIATIONS

CA - cold acclimation
CAE - cold air exposure
CWI - cold water immersion
CFI - cold finger immersion
CIVD - cold-induced vasodilation
$\dot{V}O_2$peak - peak aerobic power
$\dot{V}O_2$ - oxygen uptake
$\dot{V}CO_2$ - carbon dioxide output
HR - heart rate
Ht - height
Wt - weight
BSA - body surface area
EG - exercise group
RG - resting group
T_{re} - rectal temperature
$\bar{T}_{sk}$ - mean skin temperature
H_c - total body heat flow
TG - thermal gradient
T_{es} - esophageal temperature
I_{tissue} - tissue insulation
ECG - electrocardiogram
$\dot{M}$ - metabolic rate
BP - blood pressure
PP - pulse pressure
MAP - mean arterial pressure
$\dot{Q}$ - cardiac output
SV - stroke volume
NE - norepinephrine
SBP - systolic blood pressure
DBP - diastolic blood pressure

EXECUTIVE SUMMARY

The relative importance of core vs. skin temperature reduction as a stimulus for cold acclimation (CA) was assessed by evaluating thermoregulatory responses during cold air exposure (CAE), cold water immersion (CWI), and cold finger immersion (CFI), before and after acclimation. Subjects acclimated for 5 weeks by completing daily 1-h water immersions (20°C) while either seated quietly (RG) or performing leg exercise (EG). The exercise intensity for EG was selected to prevent core temperature from falling during each CA session. Rectal temperature fell ($P<0.05$) 0.8±0.2°C during daily immersions for RG, and did not change ($P>0.05$) for EG. CAE consisted of 40 min rest in 26°C air followed by 90 min rest in 5°C air; CWI consisted of 40 min rest in 26°C air followed by 60 min rest in 20°C water. CFI consisted of 30 min rest, then 30 min middle finger immersion in 4°C water, and was completed twice: the first trial immediately following CWI (hypothermic condition, HC); and a second trial on a separate day (normothermic condition, NC). During CAE, CWI, and CFI, there were no differences ($P>0.05$) between groups due to CA for rectal (T_{re}) or esophageal temperature (T_{es}), mean skin temperature ($\bar{T}_{sk}$), mean finger temperature, total body heat flow, tissue insulation, metabolic heat production, or cardiovascular parameters (heart rate, blood pressure, cardiac output). Thermal gradient [$TG = (T_{re} + T_{es})/2 - \bar{T}_{sk}$] during CAE was higher ($P<0.05$) after CA in RG (13.8±0.8°C) than EG (12.5±0.6°C). Norepinephrine (NE) levels increased during CAE more ($P<0.05$) post-acclimation (338±38 to 1916±288) compared to pre-acclimation (234±15 to 1184±156) for RG, but CA had no effect ($P>0.05$) on NE response for EG. During CWI, CA had no effect ($P>0.05$) on NE response between groups. During CFI, CA had no effect ($P>0.05$) on the cold-induced vasodilation (CIVD) response for either group, but CIVD was blunted ($P<0.05$) during HC, compared to NC before and after CA. In summary, the more pronounced change in NE during CAE following acclimation is consistent with increased sympathetic activation, which could mediate changes in vasomotor response to cold. The reduction of core temperature during CA sessions appears to be a necessary stimulus for this acclimation effect. This study also suggests that heat debt, not increased heat flux alone, is the stimulus for developing cold acclimation.

INTRODUCTION

Humans can be naturally or experimentally acclimated to cold (Young, 1996). The particular pattern of acclimation that develops may vary, depending on subjects' physical characteristics (Bittel, 1987), the severity of cold stress (Radomski and Boutelier, 1982; Savourey et al., 1996; Young et al., 1986), and the duration and frequency of cold exposures (Bridgman, 1991; Radomski and Boutelier, 1982; Young, 1996). An insulative pattern of cold acclimation has been demonstrated to develop following repeated daily cold water immersions (Young et al., 1986), as evidenced by a more rapid and more pronounced decrease in skin temperature upon exposure to cold air following acclimation. The authors speculated that the stimulus for acclimation was the repeated reduction in the subjects' core temperatures during each cold water immersion. However, the subjects also experienced repeated reductions in skin temperature. Thus, the relative importance of repeated reductions in skin as opposed to core temperature for the stimulation of cold acclimation was not established.

To address this question, we examined the thermoregulatory responses to cold acclimation of subjects whose core temperatures during repeated cold water immersions were either maintained constant, or allowed to decrease. Skin temperature decreased similarly in both groups during water immersion. The cold acclimation pattern was compared by evaluating physiological responses during cold air exposure and cold water immersion before and after the acclimation. It was hypothesized that repeated reduction of both core and skin temperatures would develop an insulative pattern of cold acclimation, and that such acclimation would not occur when core temperature was maintained by metabolic heat production, despite reduced skin temperature (Young, 1996).

A secondary aim was to examine the influence of core temperature change and cold acclimation on the cold-induced vasodilation (CIVD) response. Whole-body cooling has been reported to blunt or abolish CIVD (Keatinge, 1957; Rapaport et al., 1949); however, these studies did not measure core temperature. No studies have longitudinally examined the effects of whole-body cold acclimation on CIVD. Our experimental design allowed us to examine the effect of reduced core temperature as

well as effects of acclimation on CIVD. It was hypothesized that CIVD would be blunted when core temperature was reduced due to whole-body cooling, and that insulative cold acclimation would be accompanied by blunted CIVD.

METHODS

SUBJECTS

Fourteen men with no history of cold injury participated in this study, which was approved by the Institute Scientific and Human Use Review Committees. Each subject volunteered to participate after being informed of the purpose, experimental procedures, and known risks of the study. This study required 12 months to complete, and tests were evenly distributed throughout the year. The subjects who were tested during the winter months spent most of their time indoors, or dressed in cold-weather clothing while outdoors; therefore, seasonal effects were not expected to influence these experiments.

PRELIMINARY MEASUREMENTS

To assess aerobic fitness, peak oxygen uptake ($\dot{V}O_2$ peak) was determined using a continuous effort, progressive intensity cycle ergometer protocol. After a brief warm-up, subjects pedaled for 2 min at 60 rpm against zero resistance; thereafter, the power output was increased 30 W every 2 min until the subject was unable to continue in spite of verbal encouragement. Oxygen uptake ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$) were measured continuously during the exercise. $\dot{V}O_2$ peak was defined as the highest $\dot{V}O_2$ achieved. Heart rate (HR), monitored via an electrocardiogram obtained from chest electrodes (CM-5 placement) and radio telemetered to an oscilloscope-cardiotachometer, was recorded at the end of each stage.

On a separate day, the subjects' height (Ht), weight (Wt) and body composition were measured. Body density and residual lung volume were estimated by hydrostatic weighing and nitrogen dilution, respectively (Stromme et al., 1963), and these values

were used to calculate body fat content (Siri, 1961). Skinfold thickness was also measured at 10 sites: chin, subscapular, chest, side, suprailium, abdomen, triceps, thigh, knee, and calf (McArdle et al., 1984), and mean subcutaneous fat thickness was calculated (Allen et al., 1956). Body surface area (BSA) was estimated by BSA = $Wt^{0.425} \cdot Ht^{0.725} \cdot 71.84/10000$ (DuBois and DuBois, 1916).

Subjects were assigned to one of two groups, cold-water exercise (EG) or cold-water resting (RG), such that over the course of the study the groups remained of similar size and did not differ in average fitness or anthropomorphic characteristics. The physical characteristics of both groups are presented in Table 1.

Each subject in EG performed several short (8-15 min) bouts of leg exercise while immersed in 30°C water on a semirecumbent cycle ergometer modified for use in water. This was done to identify resistance eliciting 50% of $\dot{V}O_2$ peak. Fly-wheel resistance was adjusted by attaching fins of varying length to the flywheel (Shapiro et al., 1981). The seat and back of the chair were constructed of stainless steel, perforated to facilitate water movement around the subject and minimize the formation of a still boundary layer. Subjects wore aquatic sport sandles, open at the top, toes, and sides, to protect the bottom of the feet during pedaling. The ergometer was mounted on a platform that could be raised and lowered in and out of a 30,000 L pool in which water was continuously circulated and temperature maintained within ±0.1°C of the desired temperature. The water level was adjusted to cover the subjects' shoulders while they were immersed. The resistance which elicited 50% of $\dot{V}O_2$ peak was utilized for the cold-water acclimation sessions.

Table 1: Mean (±SE) physical characteristics for each group. There were no differences (P>0.05) between groups on any parameter.

Anthropometric Measurement	Resting Group (n=7)	Exercise Group (n=7)
age (yrs)	20±1	20±1
height (cm)	181±3	178±2
mass (kg)	75.5±3.2	79.8±3.2
body surface area (m^2)	1.95±0.05	1.98±0.04
mean subcutaneous fat (mm)	3.4±0.5	3.7±0.4
percent body fat (%)	16.0±0.8	15.7±1.1
$\dot{V}O_2$ peak ($mL \cdot kg^{-1} \cdot min^{-1}$)	58.2±1.8	55.4±1.6

EXPERIMENTAL DESIGN

After completing the preliminary procedures, cold air exposure (CAE) and cold water immersion (CWI) were conducted on separate days. Cold finger immersion (CFI) was conducted twice: immediately following CWI, and again on a separate day. Within 3 days after completing these tests, subjects began cold acclimation (CA). During CA, each subject was immersed to the shoulder in 20°C water for 60 min in the morning, 5 times a week for 5 weeks. During immersion, RG sat quietly, while EG performed leg exercise as described above. During alternate sessions of CA, rectal temperature (T_{re}) and HR were monitored and recorded before and every 10 min during immersion. In addition,$\dot{V}O_2$ and $\dot{V}CO_2$ were measured at min 10-20 for EG, and at min 15-25 and 45-55 for RG. In order to prevent RG from "detraining" and experiencing a decline in aerobic capacity, these subjects participated in a fitness maintenance program consisting of 60-min cycling (exercise intensity = 50% $\dot{V}O_2$ peak) on land in ambient air, twice a week during the acclimation period. The sessions for fitness maintenance program were completed 2 to 5 hours after the immersion sessions.

Within 4 days after the last CA session, CAE, CWI, and CFI were again conducted. Anthropometric characteristics and $\dot{V}O_2$ peak were also measured again. All tests were conducted at the same time of the day for each subject, between 0700 and 1100, to avoid the confounding effects of circadian rhythms. During any tests or CWA sessions, if T_{re} reached 35°C, the test or session was terminated. Subjects were instructed to fast for 12 hours before all tests.

EXPERIMENTAL PROCEDURES

Cold Air Exposure

After arriving at the laboratory, subjects voided their bladders and their nude weights were measured. For the experiment, subjects dressed in shorts and socks with holes cut over the top of the foot. They inserted a rectal thermistor 12 cm beyond the anal sphincter, and an esophageal thermocouple in the esophagus at a depth of one-fourth of the subject's height. A catheter was placed in an antecubital vein for collection of blood samples. Skin thermocouples were placed on the foot, medial thigh, chest, and back of hand. Additional skin temperature and heat flow were measured by heat flow/thermistor disks (Concept Engineering, Old Saybrook, CT) placed on the back, triceps, forearm, lateral thigh, and calf. Mean weighed skin temperature ($\bar{T}_{sk}$) was calculated as $\bar{T}_{sk} = 0.06 \cdot T_{foot} + 0.17 \cdot T_{calf} + 0.14 \cdot T_{medial\ thigh} + 0.14 \cdot T_{lateral\ thigh} + 0.14 \cdot T_{chest} + 0.07 \cdot T_{triceps} + 0.07 \cdot T_{forearm} + 0.14 \cdot T_{back} + 0.07 \cdot T_{hand}$ (Toner et al., 1984). Total body heat flow (H_c) was calculated as $H_c = 0.28\ H_{back} + 0.14\ H_{forearm} + 0.08\ H_{triceps} + 0.22\ H_{calf} + 0.28\ H_{thigh}$ (Toner et al., 1984). Thermal gradient (TG) was calculated as $TG = [(T_{re} + T_{es})/2 - \bar{T}_{sk}]$, where T_{es} is esophageal temperature. Tissue insulation (I_{tissue}) was calculated as $I_{tissue} = TG/H_c$ (Veicsteinas et al., 1982). Electrocardiogram (ECG) electrodes were attached for measurement of HR.

Subjects rested in a recumbent position for a 40 min baseline period at 26°C, then moved to a chamber where they resumed the recumbent position for 90 min exposure to 5°C air. Body temperature and heat flow data were recorded every min. Heart rate was recorded every 2 min. Metabolic rate ($\dot{M}$), normalized to body surface area, was estimated from $\dot{V}O_2$ and the respiratory exchange ratio, which were measured during baseline and for 10 min each half hour of exposure (Ravussin et al.,

1985). Blood pressure (BP) was measured before each $\dot{M}$ measurement. Pulse pressure (PP) was calculated as the difference between systolic and diastolic pressure. Mean arterial pressure (MAP) was calculated as diastolic pressure plus one third PP. Following each $\dot{M}$ measurement, cardiac output ($\dot{Q}$) was estimated in duplicate using the Collier CO_2 rebreathing technique and calculated according to the Fick principle. Stroke volume (SV) was calculated as $\dot{Q}$/HR. Blood samples were taken at the end of both baseline and exposure periods for analysis of norepinephrine (NE) by high performance liquid chromotography with electrochemical detection.

Cold Water Immersion

After arriving at the laboratory, subjects voided their bladders and their nude weights were measured. For the experiment they were dressed in swimshorts and wore aquatic sandles to protect their feet. Instrumentation and measurements were the same as indicated for CAE, above, with the exception of BP, which was only measured pre-immersion. Subjects sat quietly on the ergometer chair for a 40 min baseline period in 26°C air, after which the platform was lowered into the water (20°C) for 60 min. Immediately following the CWI, subjects were removed from the water, dried, wrapped in blankets and were instrumented for the CFI as described below.

Cold Finger Immersion

After arriving in the laboratory on the day CFI was conducted as a separate test (normothermic condition, NC), subjects voided their bladder, and inserted rectal and esophageal temperature probes. Subjects wore shirts, shorts, and shoes. On the day CFI was conducted directly after CWI (hypothermic condition, HC), subjects were wrapped in blankets, and instrumentation that was not necessary for CFI was removed. For HC, between termination of the CWI and beginning of CFI, 11.2±0.5 min elapsed. For CFI, skin thermocouples and ECG electrodes were placed on three skin sites: a) 1 cm from the tip of the right middle finger along the nail bed, b) the back of the hand, and c) the anterior forearm. The thermocouple for the finger tip was covered by approximately 1 cm^2 of Hy-Tape (Hy-Tape Surgical Products Corp., Yonkers, NY), while the other two were not covered or located over any major blood vessels. Subjects sat quietly in ambient air (26.2 ± 0.4°C) for 30 min, then immersed their middle finger to the middle phalanx into 4°C water for 30 min into a refrigerated water bath (Neslab #RTE-

111, Neslab Instruments, Inc., Newington, NH). Heart rate was recorded every 5 min. Blood pressure was measured after 25 min rest prior to finger immersion, and after 25 min of finger immersion. Body temperatures were recorded every 2 min during pre-immersion, and every 10 sec during immersion. A plastic cover supported the hand over the water bath.

STATISTICAL ANALYSIS

Data were analyzed using analysis of variance for repeated measures. Differences were evaluated by a three-way analysis among groups, trials, and times of measurement. Tukey's HSD *post-hoc* test was used to make pair-wise comparisons when a significant F-ratio was found. Statistical significance was accepted at $P<0.05$, and data reported are mean ± standard error ($\bar{X}$±SE).

A low-pass filter (SigmaPlot 3.03, Jandel Scientific, San Rafael, CA) was used on finger temperature during CFI to reduce data noise. A rise or fall in finger temperature of at least 0.5°C was considered to represent a viable CIVD. Data were examined to identify the time at which a nadir or apex occurred. The means of these data were used to construct an "average" CIVD for each of four conditions for statistical analysis: pre-acclimation normothermic, pre-acclimation hypothermic, post-acclimation normthermic, and post-acclimation hypothermic.

RESULTS

COLD ACCLIMATION

All subjects completed the full 60 min during daily immersions of CA, with the exception of one subject in RG, who was withdrawn early on his first two immersion sessions due to a fall in T_{re} to 35°C. Mean daily T_{re} immediately before immersion was similar for both groups (37.20±0.06°C). At the end of each immersion, T_{re} had fallen ($P<0.05$) to 36.35 ± 0.15°C in RG with an average daily change (ΔT_{re}) of -0.83±0.16°C. In EG, T_{re} remained unchanged ($P>0.05$) during CA sessions (37.22±0.15°C).

COLD AIR EXPOSURE

The average duration of CAE was 90±0 and 78±7 min pre-CA, and 90±0 and 87±2 min post-CA for RG and EG, respectively. Three of the subjects in EG did not complete the entire 90 min of CAE pre-CA, and two of those three did not complete the entire 90 min of CAE post-CA. All withdrawals from the exposure were reportedly due primarily to finger pain.

Figure 1 shows T_{re}, T_{es}, $\bar{T}_{sk}$, H_c, and $\dot{M}$ for CAE. Following an initial increase upon CAE, both T_{re} and T_{es} decreased by 90 min ($P<0.05$). Although H_c was higher ($P<0.05$) during CAE than pre-exposure, both H_c and $\bar{T}_{sk}$ decreased throughout CAE ($P<0.05$), and $\dot{M}$ increased ($P<0.05$). There were no differences ($P>0.05$) between groups or trials for T_{re}, $\bar{T}_{sk}$, $\dot{M}$ or H_c. At the end of cold exposure however, T_{es} was lower for RG than EG. Figure 2 shows TG and I_{tissue}. During CAE, TG increased ($P<0.05$), while I_{tissue} initially decreased ($P<0.05$) before returning to pre-exposure values. For RG, TG increased from pre- to post-acclimation. There were no differences between groups or trials for I_{tissue}. Blood pressure responses to CAE are shown in Table 2. Systolic blood pressure (SBP), diastolic blood pressure (DBP), MAP and PP all increased during CAE ($P<0.05$). Higher ($P<0.05$) SBP occurred in EG than RG, and SBP was higher pre-acclimation than post-acclimation. There were no differences between groups or trials for DBP or MAP. A higher PP was observed pre-acclimation than post-acclimation.

Figure 3 shows $\dot{Q}$, SV, HR responses to CAE. During CAE, $\dot{Q}$, SV and HR all increased ($P<0.05$). A higher $\dot{Q}$ was observed pre- than post-acclimation, with no difference between groups. There were no differences between groups or trials for SV or HR. Figure 4 shows NE, which increased with CAE ($P<0.05$). During CAE, NE was greater ($P<0.05$) for RG post-acclimation, compared to pre-acclimation, but the response did not change from pre-acclimation to post-acclimation for EG.

Figure 1: Body temperature, heat flow, and metabolic heat production ($\bar{X}\pm SE$) during Cold Air Exposure. For statistical analysis, see text. ● Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

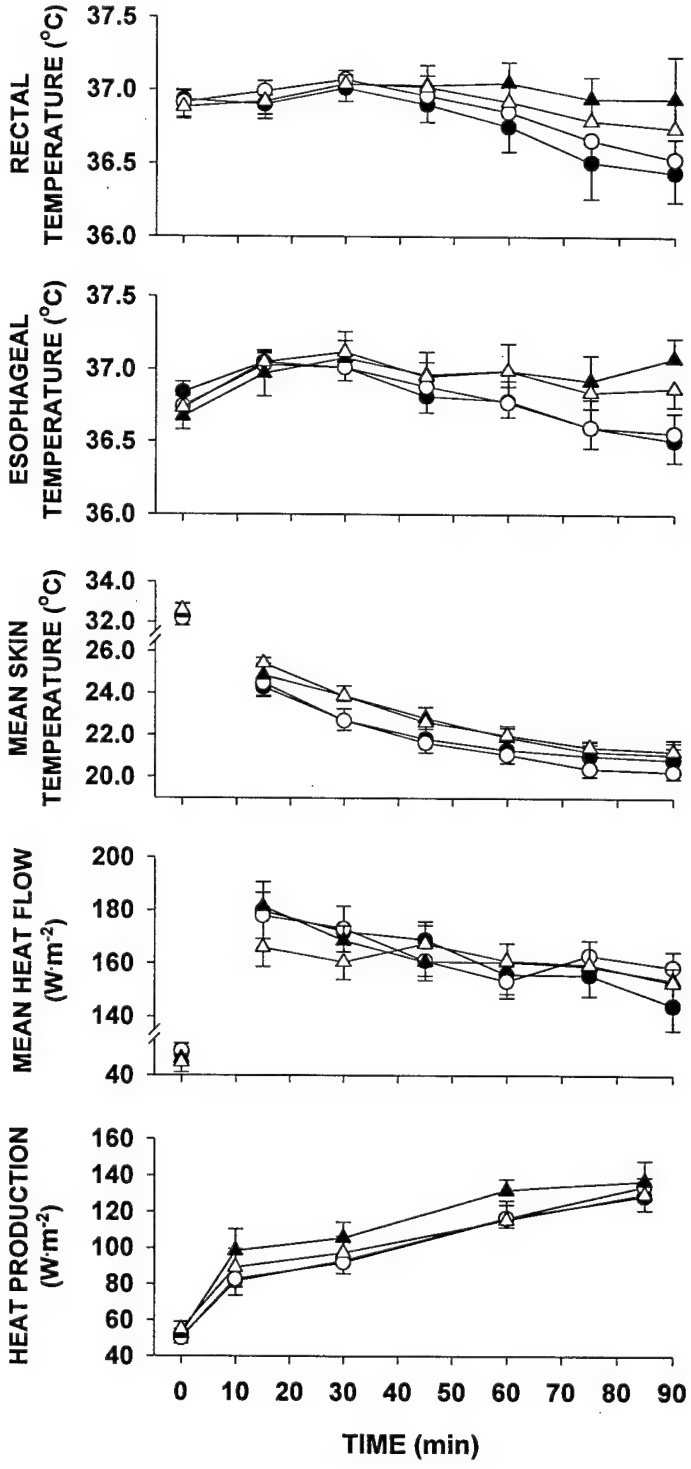


Figure 2: Thermal gradient and tissue insulation (X±SE) during Cold Air Exposure. For explanation of terms and for statistical analysis, see text. • Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

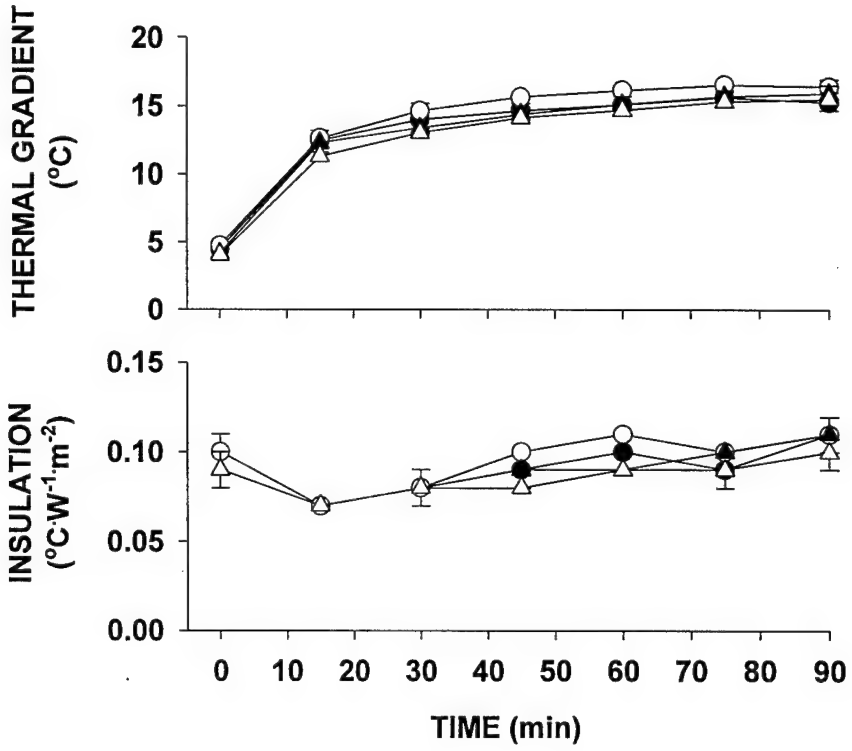


Figure 3: Cardiac output, stroke volume, heart rate (X̄±SE) during Cold Air Exposure. For statistical analysis, see text. ● Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

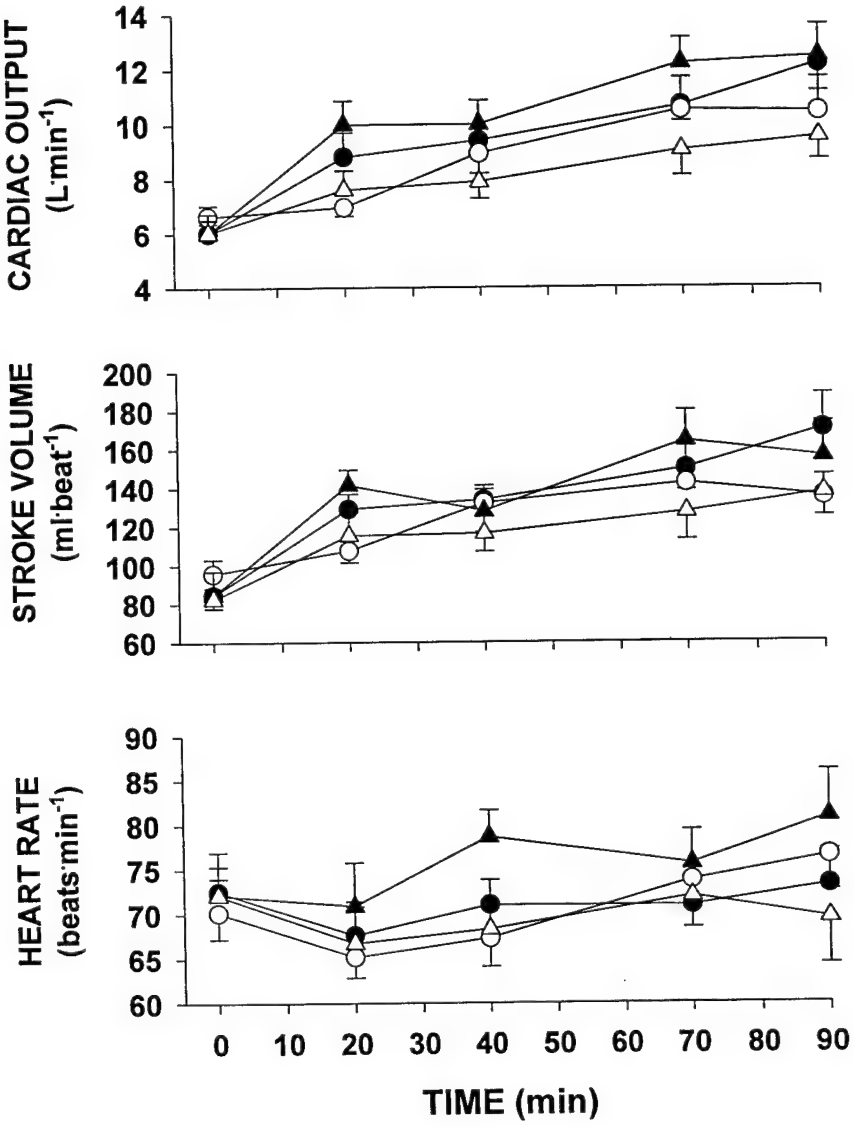


Figure 4: Norepinephrine (X±SE) during Cold Air Exposure. * Significant difference ($P<0.05$) in the norepinephrine response to cold air exposure between pre- and post-acclimation for the Resting Group.

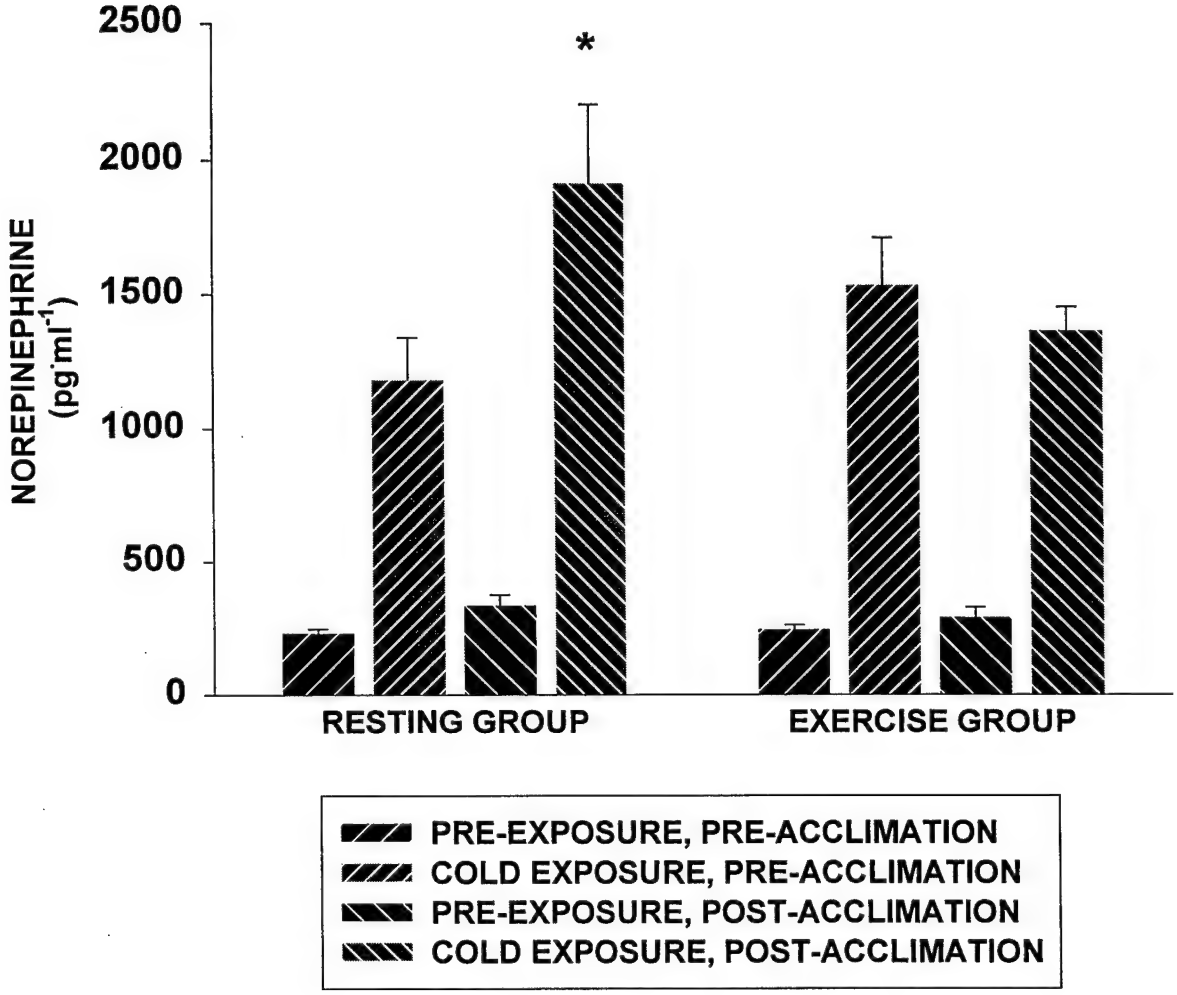


Table 2. Blood pressure ($\bar{X}$±SE, mmHg) during Cold Air Exposure. For statistical analysis, see text.

	Pre-Acclimation		Post-Acclimation	
Resting Group	Pre-exposure	Post-exposure	Pre-exposure	Post-exposure
Systolic Pressure	115±3	140±2	106±2	128±4
Diastolic Pressure	74±3	91±3	74±2	87±2
Pulse Pressure	42±3	49±3	32±3	41±4
Mean Arterial Pressure	88±2	107±2	85±1	101±2
Exercise Group				
Systolic Pressure	124±1	146±5	114±2	140±2
Diastolic Pressure	79±2	93±3	74±3	88±2
Pulse Pressure	45±2	53±2	40±4	52±2
Mean Arterial Pressure	94±2	111±4	87±2	105±2

COLD WATER IMMERSION

The average duration of CWI was 59±3 and 58±5 min pre-CA, and 60±0 and 60±1 min post-CA for RG and EG, respectively. Two subjects in RG and one subject in EG did not complete the entire 60 min of CWI pre-CA; the subject from EG also was withdrawn after 57 min post-CA. All withdraws from the immersion were due to a fall in core temperature to the medical safety limit (35°C).

Figure 5 shows T_{re}, T_{es}, H_c, and $\dot{M}$ for CWI. During CWI, T_{re} and T_{es} decreased (P<0.05). Although H_c decreased (P<0.05) during CWI, values were higher than pre-immersion (P<0.05). An increase in $\dot{M}$ occurred during CWI (P<0.05). Both T_{re} and T_{es}

exhibited a group x trial interaction ($P<0.05$), where temperatures during CWI were higher post-acclimation in EG, compared to RG. A lower ($P<0.05$) H_c occurred for EG post-acclimation, compared to pre-acclimation, but there was no change in RG. No differences were found between groups or trials for $\dot{M}$. Figure 6 shows TG and I_{tissue} responses to CWI. While TG increased upon CWI ($P<0.05$), an initial decrease ($P<0.05$) was observed in I_{tissue}. There were no differences ($P>0.05$) between groups or trials for TG. At the end of immersion, I_{tissue} was lower post-acclimation in RG, compared to pre-acclimation, but no change was observed with EG. Blood pressure prior to CWI is shown in Table 3. As with CAE, SBP was higher ($P<0.05$) for EG than RG. There was no difference between groups or trials for DBP, PP or MAP. Figure 7 shows $\dot{Q}$, SV, HR responses to CWI. While both $\dot{Q}$ and SV increased with CWI ($P<0.05$), HR decreased ($P<0.05$). A higher ($P<0.05$) $\dot{Q}$ was observed for RG than EG at the beginning of CWI, but both groups were similar by the end of immersion. A group x time interaction ($P<0.05$) showed that SV was higher for RG at the beginning of CWI, but group differences abated with time. There were no differences ($P>0.05$) in HR. Figure 8 shows the NE response to CWI. While NE increased ($P<0.05$) during CWI, no difference occurred ($P>0.05$) for either group from pre- to post-acclimation.

COLD FINGER IMMERSION

Table 4 shows BP and HR responses to CFI during NC. No changes ($P>0.05$) occurred between pre-immersion and CFI, but SBP, PP, and MAP were all higher ($P<0.05$) in EG than RG. There was no difference ($P>0.05$) between groups or trials for DBP. Figure 9, top panel shows HR responses during CFI under NC. No change ($P>0.05$) occurred in HR during CFI, but HR was higher ($P<0.05$) post-acclimation, compared to pre-acclimation. Table 5 shows BP and HR responses to CFI during HC. During CFI, SBP decreased ($P<0.05$), but there was no difference ($P>0.05$) between groups for SBP. No change occurred during CFI in DBP, PP, or MAP. Pre-acclimation DBP was higher ($P<0.05$) for EG, than RG. A group x trial interaction ($P<0.05$) shows PP was lower pre-acclimation for EG than RG, but higher post-acclimation for EG than RG. MAP was higher ($P<0.05$) in EG than RG. Figure 9, bottom panel, shows HR responses during CFI during HC. While HR increased ($P<0.05$) during CFI, there were no differences ($P>0.05$) between groups or trials.

Figure 5: Core temperature, heat flow, and metabolic heat production ($\bar{X}\pm SE$) during Cold Water Immersion. For statistical analysis, see text. ● Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

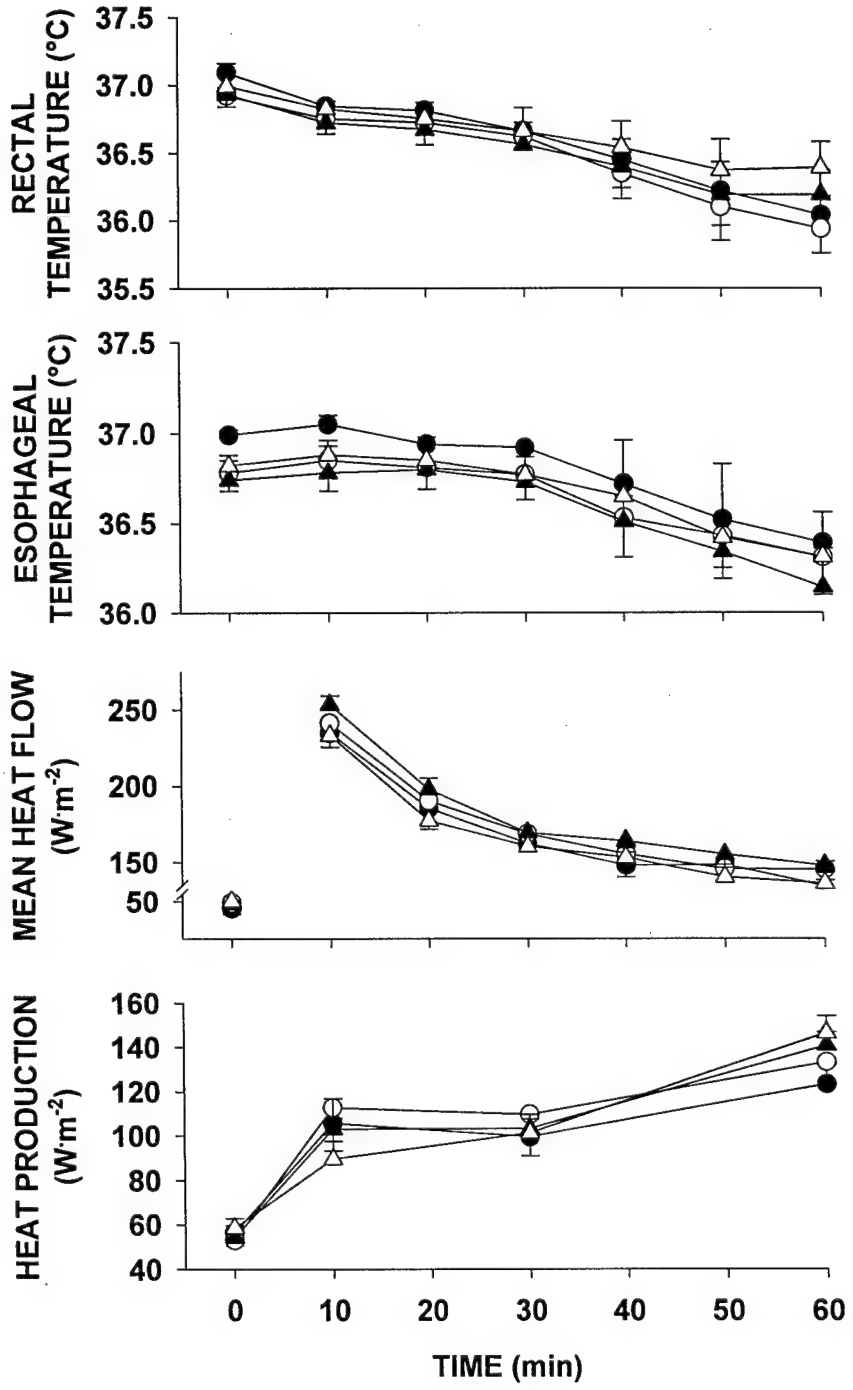


Figure 6: Thermal gradient and insulation ($\bar{X}$±SE) during Cold Water Immersion. For statistical analysis, see text. ● Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

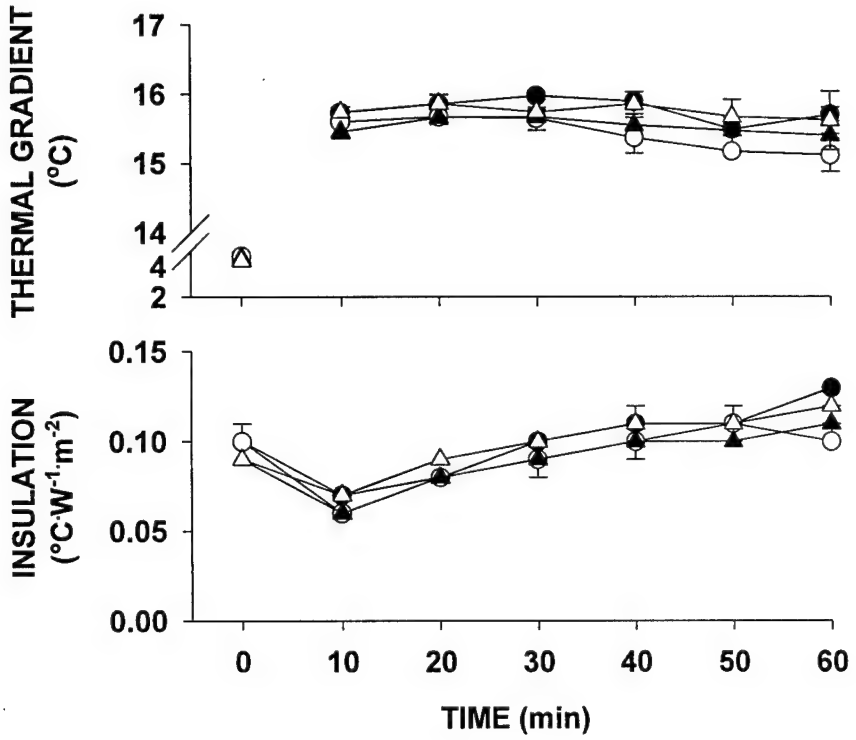


Table 3. Blood pressure ($\bar{X}$±SE, mmHg) before Cold Water Immersion. For statistical analysis, see text.

Resting Group	Pre-Acclimation	Post-Acclimation
Systolic Pressure	114±2	112±2
Diastolic Pressure	75±1	77±3
Pulse Pressure	39±2	35±4
Mean Arterial Pressure	88±1	89±2
Exercise Group		
Systolic Pressure	122±4	118±3
Diastolic Pressure	81±3	81±3
Pulse Pressure	41±2	37±4
Mean Arterial Pressure	94±3	94±2

Figure 7: Cardiac output, stroke volume, heart rate (X̄±SE) during Cold Water Immersion. For statistical analysis, see text. ● Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

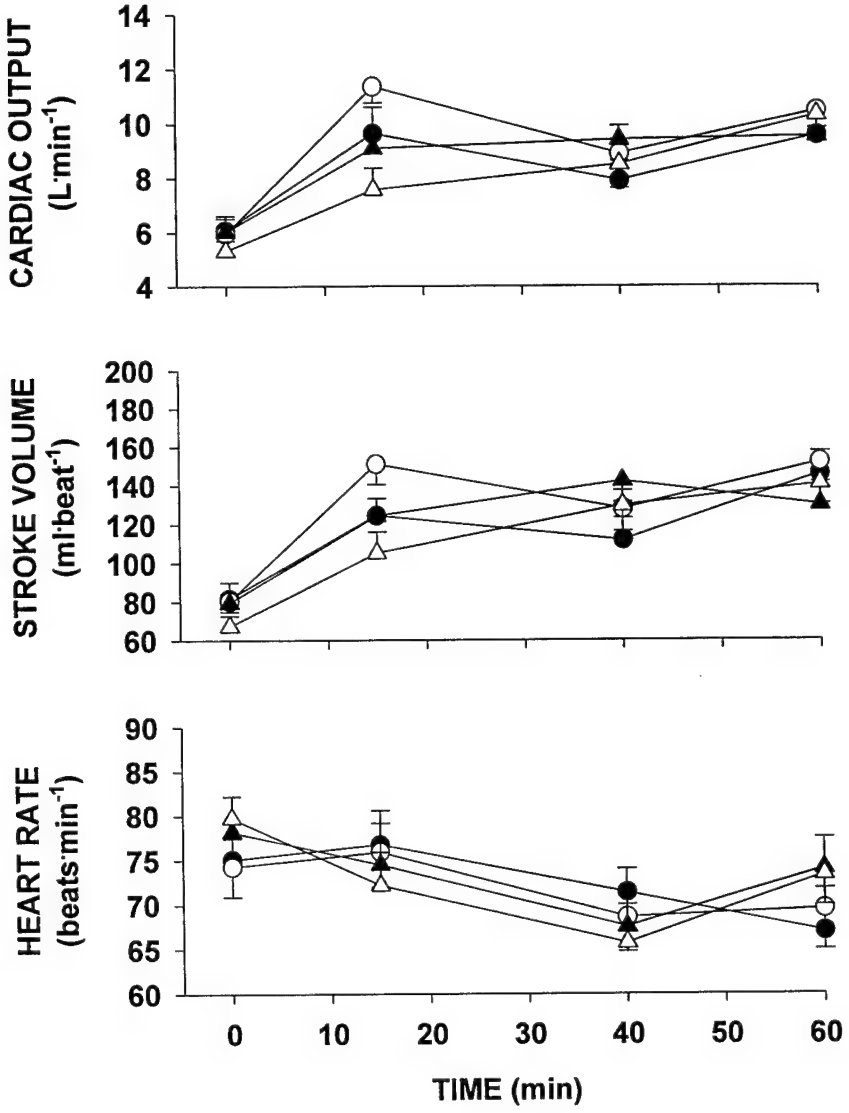


Figure 8: Norepinephrine ($\bar{X}$±SE) during Cold Water Immersion. There were no differences ($P>0.05$) between groups or trials.

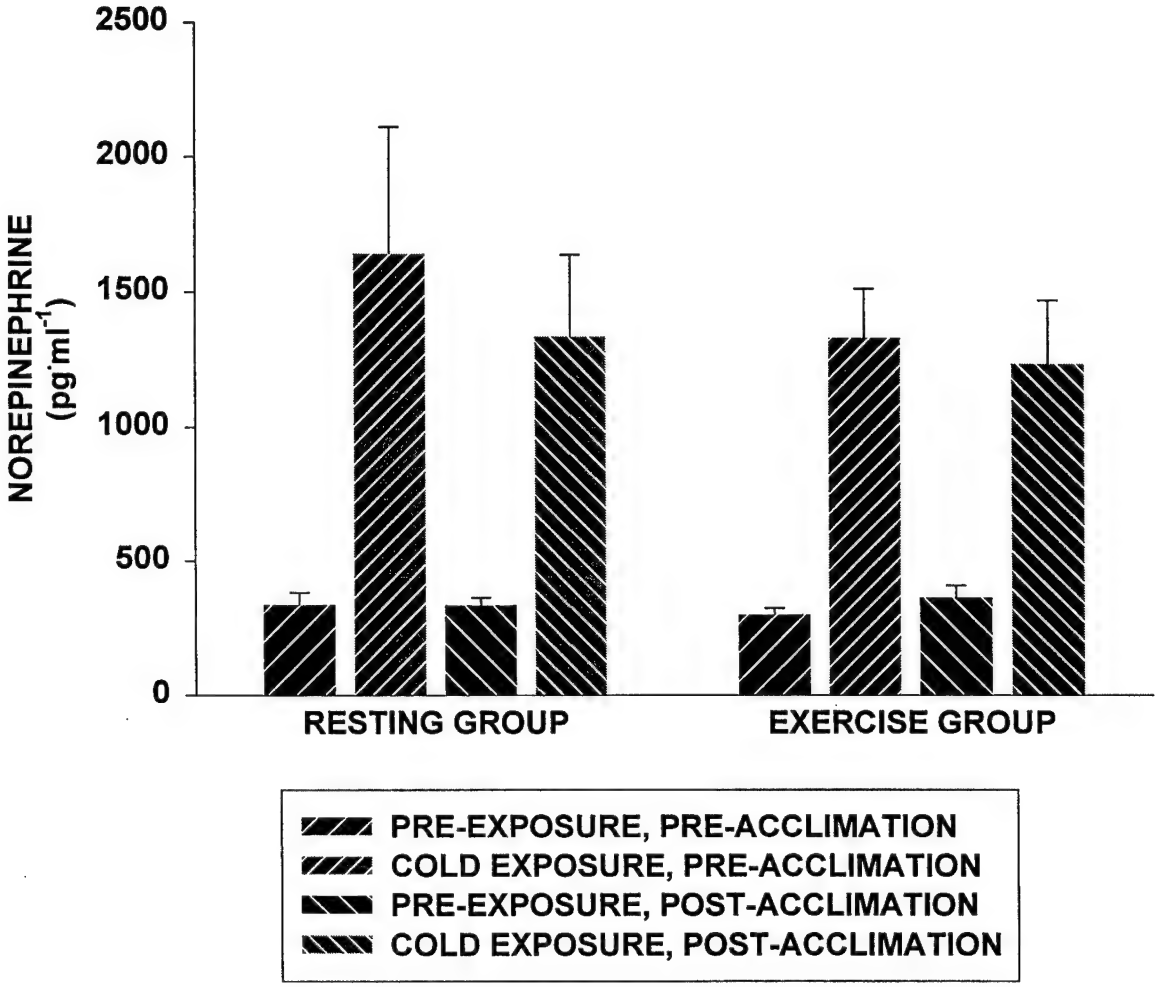


Table 4. Blood pressure ($\bar{X}$±SE, mmHg) during Cold Finger Immersion under normothermic condition. For statistical analysis, see text.

	Pre-Acclimation		Post-Acclimation	
Resting Group	Pre-immersion	25-min immersion	Pre-immersion	25-min immersion
Systolic Pressure	112±2	114±4	110±2	109±2
Diastolic Pressure	78±3	78±3	76±4	79±3
Pulse Pressure	34±2	37±3	35±3	30±4
Mean Arterial Pressure	90±3	90±3	87±2	89±2
Exercise Group				
Systolic Pressure	126±4	121±2	121±2	118±2
Diastolic Pressure	85±3	81±2	80±2	79±1
Pulse Pressure	41±4	41±3	41±2	39±3
Mean Arterial Pressure	99±3	94±2	94±2	92±1

Table 5. Blood pressure ($\bar{X}$±SE, mmHg) during Cold Finger Immersion under hypothermic condition. For statistical analysis, see text.

	Pre-Acclimation		Post-Acclimation	
Resting Group	Pre-immersion	25-min immersion	Pre-immersion	25-min immersion
Systolic Pressure	122±2	118±2	121±4	121±3
Diastolic Pressure	77±2	77±2	79±2	84±4
Pulse Pressure	45±3	40±3	42±4	37±3
Mean Arterial Pressure	92±1	91±2	93±2	96±3
Exercise Group				
Systolic Pressure	127±4	122±3	128±2	125±3
Diastolic Pressure	90±3	87±4	80±2	82±4
Pulse Pressure	37±3	35±3	48±2	44±3
Mean Arterial Pressure	102±3	99±3	96±2	96±3

Table 6 presents T_{re} and middle finger temperature (T_f) during CFI, for RG and EG, NC and HC, pre- and post-acclimation. Initial T_{re} was lower ($P<0.05$) during HC than NC, as intended by the study design. Time and temperature at the first nadir (Daanen and Hues, 1995), the first apex, and the second nadir are also presented. Only one and a half CIVD waves (nadir, apex, second nadir) included all 14 subjects during the normothermic condition, and only a single wave (first nadir and apex) included all subjects during the hypothermic condition. Initial T_f was lower ($P<0.05$) during HC than NC. During HC, T_f at the first nadir and the first apex was lower ($P<0.05$) than NC. In addition, time to the first nadir and first apex was longer ($P<0.05$) during HC than NC. No effects of acclimation were found for either RG or EG. Figure 10 shows T_f for all subjects pre-acclimation under NC and HC.

Figure 9: Heart rate during Cold Finger Immersion. Top panel is normothermic condition; bottom panel is hypothermic condition. For statistical analysis, see text. ● Resting Group, Pre-Acclimation; ○ Resting Group, Post-Acclimation; ▲ Exercise Group, Pre-Acclimation; Δ Exercise Group, Post-Acclimation.

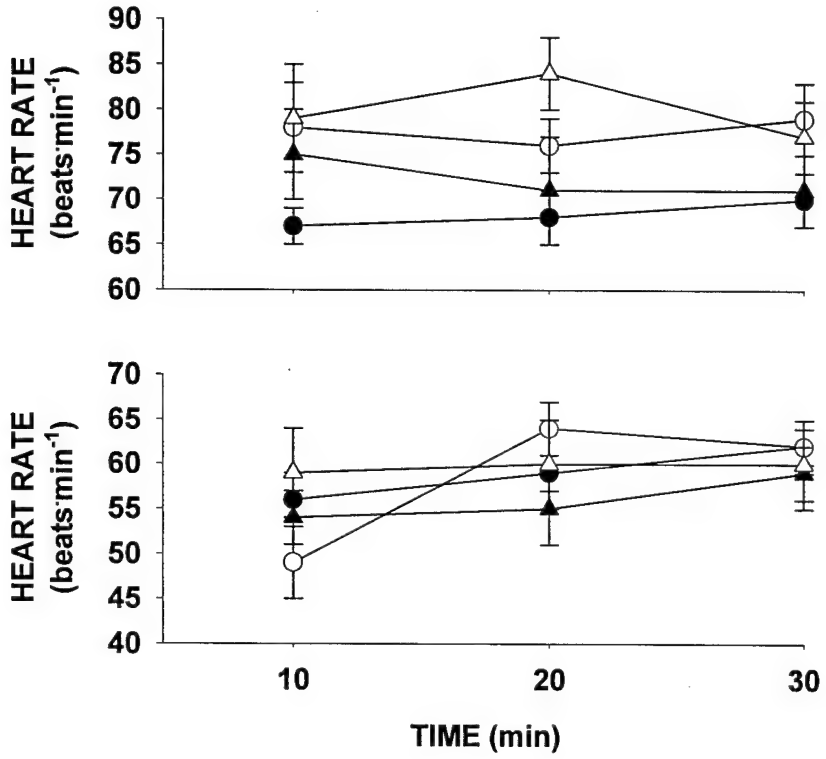
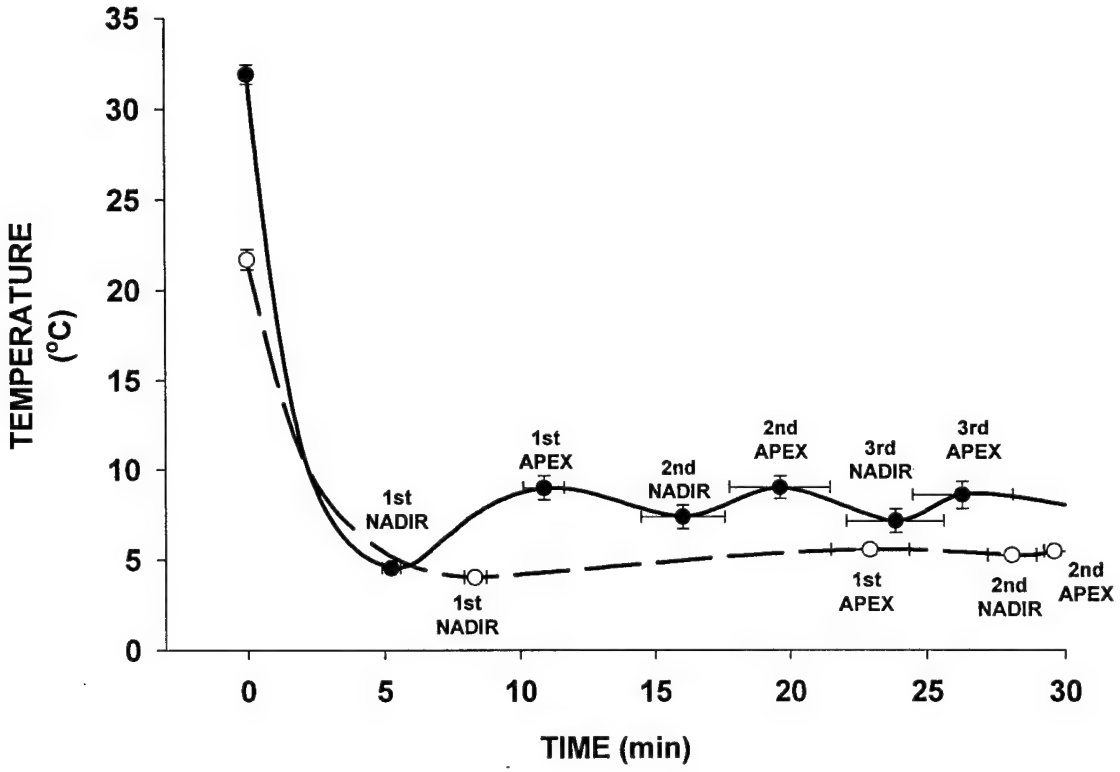


Table 6. Cold-induced vasodilation data during Cold Finger Immersion is averaged and presented ($\bar{X}$±SE) for the initial finger temperature (T_f,°C), and the time (min) and T_f at the first and second nadir and first apex. Rectal temperature (T_{re},°C) to demonstrate normothermic and hypothermic conditions. For statistical analysis, see text.

Group	Trial	Condition	Initial		First Nadir		First Apex		Second Nadir	
			T_{re}	T_f	Time	T_f	Time	T_f	Time	T_f
Resting	**Pre-acclimation**	**Normothermic**	37.03 ±0.08	32.66 ±0.54	4.95 ±0.58	4.71 ±0.19	10.67 ±1.38	9.84 ±1.00	14.64 ±2.42	8.48 ±0.94
		Hypothermic	36.03 ±0.20	21.64 ±0.84	7.86 ±0.35	4.02 ±0.03	22.02 ±1.73	5.94 ±0.36	23.83[1] ±4.15	4.70[1] ±0.80
	Post-Acclimation	**Normothermic**	37.03 ±0.07	32.24 ±0.44	4.74 ±0.18	4.79 ±0.46	10.36 ±0.81	9.92 ±0.79	18.24 ±1.56	5.45 ±0.28
		Hypothermic	36.01 ±0.20	21.74 ±0.32	7.57 ±0.38	4.02 ±0.02	21.86 ±1.96	5.83 ±0.30	28.83[1] ±0.59	5.25[1] ±0.26
Exercise	**Pre-Acclimation**	**Normothermic**	36.92 ±0.08	31.14 ±0.90	5.52 ±0.33	4.38 ±0.08	11.12 ±0.78	8.14 ±0.80	16.52 ±1.60	6.62 ±0.70
		Hypothermic	35.90 ±0.20	21.75[2] ±0.84	8.81 ±0.73	4.01 ±0.03	23.83 ±2.35	5.15 ±0.36	28.40[2] ±1.06	4.95[2] ±0.28
	Post-Acclimation	**Normothermic**	37.13 ±0.05	32.48 ±0.93	5.29 ±0.39	4.80 ±0.45	9.60 ±0.82	8.10 ±0.61	14.86 ±1.36	5.71 ±0.41
		Hypothermic	36.00 ±0.25	22.93 ±0.74	8.86 ±0.90	4.02 ±0.02	27.05 ±1.63	5.12 ±0.36	28.28[3] ±1.48	5.01[3] ±0.13

Data are presented for all 7 subjects in each group, except [1]n=6, [2]n=5, [3]n=3.

Figure 10: Finger temperatures during pre-acclimation Cold Finger Immersion under normothermic (solid line) and hypothermic (dashed line) conditions. Paired nadir or apex points (X±SE) are shown. For statistical analysis, see text.



DISCUSSION

The experimental protocol used in the present study did not stimulate development of insulative acclimation to the extent anticipated. Subjects in a previous study that demonstrated the induction of insulative cold acclimation (Young et al., 1986) were immersed daily in 18°C water for 90 min over 5 weeks, compared to 20°C for 60 min over 5 weeks in the present study. While the magnitude of the daily fall in rectal temperature for our resting group (-0.8±0.2°C) was comparable with that experienced by subjects of the previous study (-0.8±0.2°C on the first immersion, -1.0±0.3°C on the 24th immersion) (Young et al., 1986), the limited extent of acclimation in the present study suggests that duration of reduced core temperature may be an important factor for inducing insulative-cold acclimation.

The most noteworthy effect of acclimation in this study was the greater norepinephrine response to cold air exposure in the resting group following acclimation. This response has been observed in previous studies (Young et al., 1986), and its appearance in only the resting group supports the role of reduced core temperature as a necessary stimulus for insulative adaptation. During cold air exposure, the resting group also demonstrated an increase in thermal gradient following acclimation, consistent with development of insulative adapatation, although no other changes in peripheral vasoconstrictor response to cold occurred. It may be that the norepinephrine response pattern is an early adaptation, and that further changes in peripheral vasoconstrictor response would have appeared had we continued the acclimation program for a longer period of time. Both systolic blood pressure and cardiac output were lower following acclimation. While familiarization with test procedures may be a factor, this response has been observed previously (Muza et al., 1988), and a relationship between cold acclimation and control of blood pressure cannot be discounted.

No consistent pattern for cold acclimation appeared in cold water immersion data, possibly because the magnitude of cold stress during water immersion elicited a maximal response both before and after acclimation. Heat flow during the cold water immersion test was lower in the exercise group post-acclimation, compared to pre-

acclimation, while no change was observed with the resting group. This decreased heat flow was accompanied by slightly higher (P=0.08) core temperature in the exercise group post-acclimation, compared to pre-acclimation, but no change in insulation. While this response is unclear, it may suggest a role for heat flux, such as sustained by the exercise group, in acclimation to cold.

This study confirmed previous findings that whole-body cooling blunts the peripheral CIVD response (Livingstone, 1976; Keatinge, 1957). Figure 9 shows data averaged from the responses of all subjects. In these data, the variability of the time to each nadir and apex appears to be more dramatic. The variability of temperature, however, is likely limited by the cold stimulus of 4°C water. No effects of acclimation on CIVD were found for either resting or exercise groups. Previous research on the effects of acclimation are equivical, and whole-body acclimation may have a central effect resulting in a diminished CIVD response (Livingstone, 1976), whereas local cold acclimation results in more responsive vasodilation (Adams and Smith, 1962). Thus alterations in the CIVD response may be related to central thermal state, rather than to local finger temperature (Adams and Smith, 1962). It may be that the minimal development of acclimation in this experiment did not allow acclimation effects on CIVD to appear.

In conclusion, several new findings are apparent from the results of this study. A decrease in core temperature appears to be a necessary stimulus for the development of insulative adaptation. In addition, a minimum duration of reduction in core temperature may also be required. The relative importance of each of these factors remains to be determined. From the results of this study and previous work (Young et al., 1986), a cold water acclimation program should incorporate a core temperature decrease of 0.8-1.0°C for 90 min, 5 times per week for 5 weeks, in order to provide a minimum stimulus for the development of insulative adaptation. An early manifestation of this adaptation is an increased norepinephrine response to cold air exposure. Future research should consider the benefit of periodic evaluations throughout the course of acclimation in order to better characterize the development of the adaptation.

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